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     FILE 'BIOSIS, MEDLINE, SCISEARCH, CA' ENTERED AT 09:57:35 ON 22 AUG 2002
L1          534 S TRADD
L2        84555 S ANTISENSE OR ANTI-SENSE OR (COMPLEMENTA? (2N) OLIGONUCL?)
L3           20 S L1 AND L2
L4           11 DUP REM L3 (9 DUPLICATES REMOVED)
L5          609 S MONIA, B?/AU
L6          352 S COWSERT, L?/AU
L7          702 S (L5 OR L6) AND L2
L8            2 S L7 AND L1

=> d l4 1-11 ibib abs; d l8 1-2 ibib abs

L4   ANSWER 1 OF 11  CA  COPYRIGHT 2002 ACS
ACCESSION NUMBER:        134:348630  CA
TITLE:                   New members of the TRAF (tumor necrosis factor
                         receptor-associated factor) protein family with
                         possible therapeutic uses
INVENTOR(S):             Zapata, Juan M.; Reed, John C.
PATENT ASSIGNEE(S):      The Burnham Institute, USA
SOURCE:                  PCT Int. Appl., 156 pp.
                         CODEN: PIXXD2
DOCUMENT TYPE:           Patent
LANGUAGE:                English
FAMILY ACC. NUM. COUNT:  1
PATENT INFORMATION:

     PATENT NO.        KIND  DATE          APPLICATION NO.  DATE
     ----------------  ----  --------      ---------------  --------
     WO 2001032696      A2   20010510      WO 2000-US30533  20001103
     WO 2001032696      A3   20020117
         W:  AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH,
             CN, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EE, EE, ES, FI, FI,
             GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR,
             KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX,
             MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM,
             TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ,
             MD, RU, TJ, TM
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
             DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
             BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
     EP 1228088         A2   20020807      EP 2000-975594   20001103
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
PRIORITY APPLN. INFO.:                  US 1999-434784   A2 19991105
                                        WO 2000-US30533  W  20001103
AB   In accordance with the present invention, there are provided novel
     TRAF-Protein-Binding-Domain polypeptides (TPBDs). The invention also
     provides nucleic acid mols. encoding TPBDs, vectors contg. these nucleic
     acid mols. and host cells contg. the vectors. The invention also provides
     antibodies that can specifically bind to invention TPBDs. Such TPBDs
     and/or anti-TPBD antibodies are useful for discovery of drugs that
     suppress autoimmunity, inflammation, allergy, allograft rejection, sepsis,
     and other diseases. Characterization of the proteins is reported and
     their interaction of other members of the family. A reporter gene assay
     for measuring their effects on NF-.kappa.B activity is described.

L4   ANSWER 2 OF 11  CA  COPYRIGHT 2002 ACS
ACCESSION NUMBER:        134:96296  CA
TITLE:                   (Sequences of novel internal ribosome entry sites
                         (IRES) of human and mouse X-linked inhibitor of
                         apoptosis (XIAP) and uses thereof in modulating
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cap-independent translation
INVENTOR(S): Korneluk, Robert G.; Holcik, Martin; Liston, Peter
PATENT ASSIGNEE(S): Apoptogen, Inc., Can.
SOURCE: U.S., 35 pp., Cont.-in-part of U.S. Ser. No. 121,979.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6171821	B1	20010109	US 1999-332319	19990614
US 6159709	A	20001212	US 1998-121979	19980724
WO 2000005366	A2	20000203	WO 1999-IB1415	19990722
WO 2000005366	A3	20000615		
W: CA, JP, US				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
EP 1100900	A2	20010523	EP 1999-935002	19990722
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRIORITY APPLN. INFO.: US 1998-121979 A2 19980724
US 1999-332319 A2 19990614
WO 1999-IB1415 W 19990722

AB The invention features purified nucleic acid encoding a novel internal ribosome entry site (IRES) sequence from the human and mouse X-linked inhibitor of apoptosis (XIAP) gene. The invention also features methods for using the XIAP IRES to increase cap-independent translation of polypeptide coding sequences linked to the XIAP IRES, and methods for isolating compds. that modulate cap-independent translation.

REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 3 OF 11 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 1
ACCESSION NUMBER: 2001:219574 BIOSIS
DOCUMENT NUMBER: PREV200100219574
TITLE: Hyaluronidase induction of a WW domain-containing oxidoreductase that enhances tumor necrosis factor cytotoxicity.
AUTHOR(S): Chang, Nan-Shan (1); Pratt, Nicole; Heath, John; Schultz, Lori; Sleve, Daniel; Carey, Gregory B.; Zevotek, Nicole
CORPORATE SOURCE: (1) Lab. of Molecular Immunology, Guthrie Research Inst., 1 Guthrie Square, Sayre, PA, 18840: nschang@inet.guthrie.org USA
SOURCE: Journal of Biological Chemistry, (February 2, 2001) Vol. 276, No. 5, pp. 3361-3370. print.
ISSN: 0021-9258.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English

AB To determine how hyaluronidase increases certain cancer cell sensitivity to tumor necrosis factor (TNF) cytotoxicity, we report here the isolation and characterization of a hyaluronidase-induced murine WW domain-containing oxidoreductase (WOX1). WOX1 is composed of two N-terminal WW domains, a nuclear localization sequence, and a C-terminal alcohol dehydrogenase (ADH) domain. WOX1 is mainly located in the mitochondria, and the mitochondrial targeting sequence was mapped within the ADH domain. Induction of mitochondrial permeability transition by TNF, staurosporine, and atractyloside resulted in WOX1 release from mitochondria and subsequent nuclear translocation. TNF-mediated WOX1

nuclear translocation occurred shortly after that of nuclear factor-kappaB nuclear translocation, whereas both were independent events. WOX1 enhanced TNF cytotoxicity in L929 cells via its WW and ADH domains as determined using stable cell transfectants. In parallel with this observation, WOX1 also enhanced **TRADD** (TNF receptor-associated death domain protein)-mediated cell death in transient expression experiments. **Antisense** expression of WOX1 raised TNF resistance in L929 cells. Enhancement of TNF cytotoxicity by WOX1 is due, in part, to its significant down-regulation of the apoptosis inhibitors Bcl-2 and Bcl-xL (>85%), but up-regulation of pro-apoptotic p53 (apprx200%) by the ADH domain. When overexpressed, the ADH domain mediated apoptosis, probably due to modulation of expression of these proteins. The WW domains failed to modulate the expression of these proteins, but sensitized COS-7 cells to TNF killing and mediated apoptosis in various cancer cells independently of caspases. Transient cotransfection of cells with both p53 and WOX1 induced apoptosis in a synergistic manner. WOX1 colocalizes with p53 in the cytosol and binds to the proline-rich region of p53 via its WW domains. Blocking of WOX1 expression by **antisense** mRNA abolished p53 apoptosis. Thus, WOX1 is a mitochondrial apoptogenic protein and an essential partner of p53 in cell death.

L4 ANSWER 4 OF 11 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2001:357789 BIOSIS
DOCUMENT NUMBER: PREV200100357789
TITLE: Constitutive activation of NFkappaB prevents TRAIL-induced apoptosis in renal cancer cells.
AUTHOR(S): Oya, Mototsugu (1); Ohtsubo, Masafumi (1); Takayanagi, Atsushi (1); Shimizu, Nobuyoshi (1); Murai, Masaru (1)
CORPORATE SOURCE: (1) Tokyo Japan
SOURCE: Journal of Urology, (May, 2001) Vol. 165, No. 5 Supplement, pp. 120. print.
Meeting Info.: Annual Meeting of the American Urological Association, Inc. Anaheim, California, USA June 02-07, 2001
ISSN: 0022-5347.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English

L4 ANSWER 5 OF 11 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2001:158717 BIOSIS
DOCUMENT NUMBER: PREV200100158717
TITLE: **Antisense** modulation of **TRADD** expression.
AUTHOR(S): Monia, Brett P.; Cowsert, Lex M.
ASSIGNEE: Isis Pharmaceuticals Inc.
PATENT INFORMATION: US 6077672 June 20, 2000
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (June 20, 2000) Vol. 1235, No. 3, pp. No Pagination. e-file.
ISSN: 0098-1133.
DOCUMENT TYPE: Patent
LANGUAGE: English
AB **Antisense** compounds, compositions and methods are provided for modulating the expression of **TRADD**. The compositions comprise **antisense** compounds, particularly **antisense** oligonucleotides, targeted to nucleic acids encoding **TRADD**. Methods of using these compounds for modulation of **TRADD** expression and for treatment of diseases associated with expression of **TRADD** are provided.

L4 ANSWER 6 OF 11 CA COPYRIGHT 2002 ACS
ACCESSION NUMBER: 132:203178 CA

TITLE: **Antisense** modulation of **TRADD** expression
INVENTOR(S): Monia, Brett P.; Cowsert, Lex M.
PATENT ASSIGNEE(S): Isis Pharmaceuticals, Inc., USA
SOURCE: PCT Int. Appl., 88 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000012527	A1	20000309	WO 1999-US19614	19990825
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 6077672	A	20000620	US 1998-143212	19980828
AU 9955875	A1	20000321	AU 1999-55875	19990825

PRIORITY APPLN. INFO.: US 1998-143212 A 19980828
WO 1999-US19614 W 19990825

AB **Antisense** compds., compns. and methods are provided for modulating the expression of **TRADD**. The compns. comprise **antisense** compds., particularly **antisense** oligonucleotides, targeted to nucleic acids encoding **TRADD**. Methods of using these compds. for modulation of **TRADD** expression and for treatment of diseases assocd. with expression of **TRADD** are provided.

REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 7 OF 11 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 2
ACCESSION NUMBER: 2000:468362 BIOSIS
DOCUMENT NUMBER: PREV200000468362
TITLE: Mechanism of chronic obstructive uropathy: Increased expression of apoptosis-promoting molecules.
AUTHOR(S): Choi, Yeong-Jin; Baranowska-Daca, Elzbieta; Nguyen, Vinh; Koji, Takehiko; Ballantyne, Christie M.; Sheikh-Hamad, David; Suki, Wadi N.; Truong, Luan D. (1)
CORPORATE SOURCE: (1) Department of Pathology, Methodist Hospital, 6565 Fannin, Houston, TX, 77030 USA
SOURCE: Kidney International, (October, 2000) Vol. 58, No. 4, pp. 1481-1491. print.
ISSN: 0085-2538.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Background: We have demonstrated that renal tubular and interstitial cells undergo pronounced apoptosis during the course of chronic obstructive uropathy (COU). Apoptosis is a complex cellular process consisting of multiple steps, each of which is mediated by families of related molecules. These families may include receptor/ligand molecules such as Fas, Fas ligand, tumor necrosis factor receptor-1 (TNFR-1), and TNF-related apoptosis inducing ligand (TRAIL); signal transduction adapter molecules such as Fas-associated death domain (FADD), TNFR-1 associated

death domain (**TRADD**), receptor-interacting protein (RIP), Fas-associated factor (FAF), and Fas-associated phosphatase (FAP); or effector molecules such as caspases. However, the mechanism of tubular cell apoptosis, as well as the pathogenetic relevance of these apoptosis-related molecules in COU, remains poorly understood. Methods: Kidneys were harvested from sham-operated control mice and mice with COU created by left ureter ligation sacrificed in groups of three at days 4, 15, 30, and 45. To detect apoptotic tubular and interstitial cells, in situ end labeling of fragmented DNA was performed. To detect the expression of apoptosis-related molecules, ribonuclease protection assay was used with specific **antisense** RNA probes for Fas, Fas ligand, TNFR-1, TRAIL, FADD, **TRADD**, RIP, FAF, FAP, and caspase-8. Immunostaining for Fas, Fas ligand, TRAIL, **TRADD**, RIP, and caspase-8 was also performed. To assess the role of these molecules in COU-associated renal cell apoptosis, the frequencies of apoptotic tubular and interstitial cells were separately quantitated for each experimental time point, and their patterns of variation were correlated with those of apoptosis-related molecules. Results: The obstructed kidneys displayed increased apoptosis of both tubular and interstitial cells. Tubular cell apoptosis appeared at day 4 after ureter ligation, peaked (fivefold of control) at day 15, and decreased gradually until the end of the experiment. In contrast, interstitial cell apoptosis sustained a progressive increase throughout the experiment. Apoptosis was minimal at all experimental time points for control and contralateral kidneys. Compared with control and contralateral kidneys, the ligated kidneys displayed a dynamic expression of mRNAs for many apoptosis-related molecules, which included an up to threefold increase for Fas, Fas ligand, TNF-R1, TRAIL, **TRADD**, RIP, and caspase-8, and an up to twofold increase for FADD and FAP, but there was little change for FAF. These mRNAs increased between days 4 and 15, decreased until day 30, but then increased again until day 45. The rise and fall of mRNAs between days 4 and 30 paralleled a similar fluctuation in tubular cell apoptosis in that period. The subsequent increase of mRNAs was correlated with a continuous rise of interstitial cell apoptosis. We demonstrated a positive immunostaining for Fas and Fas ligand in the tubular cells at early time points as well as in interstitial inflammatory cells at later time points. Although increased expression of TRAIL, **TRADD**, RIP, and caspase-8 was noted in tubular cells, there was no staining for these molecules in interstitial cells. Conclusion: The current study documents a dynamic expression of several molecules that are known to mediate the most crucial steps of apoptosis. It implicates these molecules in COU-associated renal cell apoptosis and in the pathogenesis of this condition. It also lays the foundation for interventional studies, including genetic engineering, to evaluate the molecular control of apoptosis associated with COU.

L4 ANSWER 8 OF 11 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 3

ACCESSION NUMBER: 1999:310607 BIOSIS
DOCUMENT NUMBER: PREV199900310607
TITLE: The interaction of p62 with RIP links the atypical PKCs to NF-kappaB activation.
AUTHOR(S): Sanz, Laura; Sanchez, Pilar; Lallena, Maria-Jose; Diaz-Meco, Maria T.; Moscat, Jorge (1)
CORPORATE SOURCE: (1) Laboratorio Glaxo Wellcome-CSIC de Biologia Molecular y Celular, Centro de Biologia Molecular 'Severo Ochoa' Consejo Superior de Investigaciones Cientificas, Universidad Autonoma de Madrid, Universidad Autonoma, Canto Blanco, 28049, Madrid Spain
SOURCE: EMBO (European Molecular Biology Organization) Journal, (June 1, 1999) Vol. 18, No. 11, pp. 3044-3053. ISSN: 0261-4189.

DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English

AB The two members of the atypical protein kinase C (aPKC) subfamily of isozymes (zetaPKC and lambda/iotaPKC) are involved in the control of nuclear factor kappaB (NF-kappaB) through IKKbeta activation. Here we show that the previously described aPKC-binding protein, p62, selectively interacts with RIP but not with TRAF2 in vitro and in vivo. p62 bridges the aPKCs to RIP, whereas the aPKCs link IKKbeta to p62. In this way, a signaling cascade of interactions is established from the TNF-R1 involving TRADD/RIP/p62/aPKCs/IKKbeta. These observations define a novel pathway for the activation of NF-kappaB involving the aPKCs and p62. Consistent with this model, the expression of a dominant-negative mutant lambda/iotaPKC impairs RIP-stimulated NF-kappaB activation. In addition, the expression of either an N-terminal aPKC-binding domain of p62, or its C-terminal RIP-binding region are sufficient to block NF-kappaB activation. Furthermore, transfection of an antisense construct of p62 severely abrogates NF-kappaB activation. Together, these results demonstrate that the interaction of p62 with RIP serves to link the atypical PKCs to the activation of NF-kappaB by the TNFalpha signaling pathway.

L4 ANSWER 9 OF 11 CA COPYRIGHT 2002 ACS
ACCESSION NUMBER: 129:256015 CA
TITLE: Receptor-interacting protein-associated protein (RAP), its cDNA, and RAP-related modulators of RIP proteins for use as pharmaceuticals
INVENTOR(S): Wallach, David; Kovalenko, Andrei
PATENT ASSIGNEE(S): Yeda Research and Development Co. Ltd., Israel
SOURCE: PCT Int. Appl., 65 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9841624	A1	19980924	WO 1998-IL125	19980319
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9866347	A1	19981012	AU 1998-66347	19980319
AU 747005	B2	20020509		
EP 972033	A1	20000119	EP 1998-908273	19980319
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
BR 9808915	A	20000801	BR 1998-8915	19980319
JP 2001519656	T2	20011023	JP 1998-540291	19980319
NO 9904524	A	19991111	NO 1999-4524	19990917
US 2002058024	A1	20020516	US 2001-927458	20010813

PRIORITY APPLN. INFO.: IL 1997-120485 A 19970319
WO 1998-IL125 W 19980319
US 1999-381358 A2 19990920

AB The cDNA for the title RAP protein and the RAP protein are disclosed. Modulators of RIP biol. activity and their pharmaceutical uses, such as treatment of tumors or HIV-infected cells, are also disclosed. The RAP

protein cDNA was identified using a two-hybrid assay. Binding studies indicated that RAP essentially binds only to RIP and does not bind to **TRADD**, MORT-1, p55-R, p75-R or MACH. Other studies showed that RAP did not protect cells from tumor necrosis factor killing but does block NF-.kappa.B activation by **TRADD**, RIP and p55 tumor necrosis factor receptor and does block Jun kinase induction by RIP.

L4 ANSWER 10 OF 11 CA COPYRIGHT 2002 ACS
ACCESSION NUMBER: 126:211035 CA
TITLE: MACH proteins and cDNAs and method for modulating tumor necrosis factor receptor and FAS receptor signaling
INVENTOR(S): Wallach, David; Boldin, Mark; Goncharov, Tanya; Goltsev, Yury V.
PATENT ASSIGNEE(S): Yeda Research and Development Co. Ltd., Israel; Weinwurzel, Henry; Wallach, David; Boldin, Mark; Goncharov, Tanya; Goltsev, Yury V.
SOURCE: PCT Int. Appl., 163 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9703998	A1	19970206	WO 1996-US10521	19960614
W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG				
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA				
AU 9661805	A1	19970218	AU 1996-61805	19960614
AU 708799	B2	19990812		
CN 1198165	A	19981104	CN 1996-196658	19960614
EP 914325	A1	19990512	EP 1996-919472	19960614
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI				
JP 11509422	T2	19990824	JP 1996-506675	19960614
NO 9800198	A	19980309	NO 1998-198	19980115
US 6399327	B1	20020604	US 1998-983502	19980410

PRIORITY APPLN. INFO.:
IL 1995-114615 A 19950716
IL 1995-114986 A 19950817
IL 1995-115319 A 19950914
IL 1995-116588 A 19951227
IL 1996-117932 A 19960416
WO 1996-US10521 W 19960614

AB The present invention provides proteins capable of modulating or mediating the FAS receptor ligand or TNF effect on cells carrying FAS receptor or p55 receptor by binding or interacting with MORT-1 protein, which in turn binds to the intracellular domain of the FAS receptor or to another protein **TRADD** which binds to the p55 receptor. In addn., peptide inhibitors which interfere with the proteolytic activity of MORT-1-binding proteins having proteolytic activity are provided as well as a method of designing them. The cDNAs for isoforms .alpha.1, .alpha.2, .alpha.3, .beta.1, .beta.2, .beta.3, .beta.4 and .beta.5 of the MORT1-assocd. CED3 homolog (MACH protein) of human cells were cloned and sequenced. The C-terminal region of the .alpha.1, .alpha.2 and .alpha.3 isoforms exhibit sequence homol. with CED3/ICE proteases. These domains of the .alpha. isoforms were shown to have protease activity. MACH.alpha.1 and MACH.beta.1 were coimmunopptd. with MORT-1 from lysates

of human embryonic kidney 293-EBNA cells. Direct interaction of MACH.alpha.1 and MACH.beta.1 was also demonstrated. Blocking of MACH.alpha. function was found to interfere with cell death induction by FAS and tumor necrosis factor receptors.

L4 ANSWER 11 OF 11 CA COPYRIGHT 2002 ACS
ACCESSION NUMBER: 125:266049 CA
TITLE: Human death-domain-motif-contg. proteins and their modulators, recombinant methods, and treatment of virus infection or tumor
INVENTOR(S): Wallach, David; Boldin, Mark P.; Varfolomeev, Eugene E.; Pancer, Zeev; Mett, Igor; Goncharov, Tanya M.
PATENT ASSIGNEE(S): Yeda Research and Development Co. Ltd., Israel
SOURCE: PCT Int. Appl., 72 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9625941	A1	19960829	WO 1996-US2326	19960215
W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI				
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE				
CA 2213484	AA	19960829	CA 1996-2213484	19960215
AU 9651332	A1	19960911	AU 1996-51332	19960215
EP 813419	A1	19971229	EP 1996-907886	19960215
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV				
JP 11500622	T2	19990119	JP 1996-525791	19960215
ZA 9601415	A	19960826	ZA 1996-1415	19960222
US 6355780	B1	20020312	US 1997-894626	19971209

PRIORITY APPLN. INFO.: IL 1995-112742 A 19950222
IL 1995-115289 A 19950913
WO 1996-US2326 W 19960215

AB A modulator of regulatory cellular events occurring intracellularly which are mediated by regulatory proteins contg. a "death domain" motif is provided the "death domain" is a regulatory portion of the regulatory proteins, and the modulator is capable of interacting with one or more "death domain" motifs contained in the regulatory proteins and affecting the regulatory action of one or more of the regulatory proteins. The modulator preferably is capable of interacting with "death domain" motifs within p55-TNF-R, FAS/APO1-R, NGF-R, MORT-1, RIP, **TRADD**, or ankyrin. A method for producing the modulators is also provided. The modulators are useful for modulating functions mediated in cells by proteins contg. the "death domain".

L8 ANSWER 1 OF 2 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2001:158717 BIOSIS
DOCUMENT NUMBER: PREV200100158717
TITLE: **Antisense** modulation of **TRADD** expression.
AUTHOR(S): **Monia, Brett P.; Cowsert, Lex M.**
ASSIGNEE: Isis Pharmaceuticals Inc.

PATENT INFORMATION: US 6077672 June 20, 2000
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (June 20, 2000) Vol. 1235, No. 3, pp. No Pagination. e-file.
ISSN: 0098-1133.
DOCUMENT TYPE: Patent
LANGUAGE: English

AB **Antisense** compounds, compositions and methods are provided for modulating the expression of **TRADD**. The compositions comprise **antisense** compounds, particularly **antisense** oligonucleotides, targeted to nucleic acids encoding **TRADD**. Methods of using these compounds for modulation of **TRADD** expression and for treatment of diseases associated with expression of **TRADD** are provided.

L8 ANSWER 2 OF 2 CA COPYRIGHT 2002 ACS
ACCESSION NUMBER: 132:203178 CA
TITLE: **Antisense** modulation of **TRADD** expression
INVENTOR(S): **Monia, Brett P.; Cowsert, Lex M.**
PATENT ASSIGNEE(S): Isis Pharmaceuticals, Inc., USA
SOURCE: PCT Int. Appl., 88 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000012527	A1	20000309	WO 1999-US19614	19990825
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 6077672	A	20000620	US 1998-143212	19980828
AU 9955875	A1	20000321	AU 1999-55875	19990825

PRIORITY APPLN. INFO.: US 1998-143212 A 19980828
WO 1999-US19614 W 19990825

AB **Antisense** compds., compns. and methods are provided for modulating the expression of **TRADD**. The compns. comprise **antisense** compds., particularly **antisense** oligonucleotides, targeted to nucleic acids encoding **TRADD**. Methods of using these compds. for modulation of **TRADD** expression and for treatment of diseases assocd. with expression of **TRADD** are provided.

REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT